State Department of Public Health to effect regulatory measures to stop the spread of this disease were met with such strenuous opposition from a majority of the aviary owners, many of whom do not recognize the existence of psittacosis, that little could be accomplished in the way of control measures.

Up to this time—October, 1932—there were fifty-four cases with ten deaths in this state, and outbreaks in eight other states as follows, all traced to California parrakeets:

New York, November, 1931	3 cases	1 death
Oregon, November, 1931	2 cases (5 suspects)	2 deaths
Illinois, July, 1932	3 cases	1 death
Michigan, August, 1932	1 case	1 death
Minnesota, September, 1932	12 cases	1 death
Massachusetts, October, 1932	2 cases	1 death
Wisconsin, October, 1932	9 cases	0 deaths
Idaho, October, 1932	1 case	recovered

—and that one case happened to be the wife of United States Senator Borah.

Then things began to happen in Washington. The Surgeon-General issued an order prohibiting the interstate transportation by common carrier of birds of the parrot family, and provided that they could be released for interstate shipment only after being certified by the State Department of Public Health. Very few could be certified and a quarantine was placed on all others; consequently most of the traffic in parrakeets ceased.

As the industry was practically tied up under federal and state regulations, the aviary owners assumed a more coöperative attitude, and definite regulatory measures were instituted.

Breeders were required to maintain at least three pens separated by a distance of five feet. The first pen was maintained for breeding purposes only; the second for maturing the birds to the age of seven months, at which time they were given a leg-band on which was stamped the registration number and code number of the owner, and placed in the third isolation pen for a period of thirty days. At the end of this period, the birds were inspected and a certificate of health given for their release. This procedure seemed very effective as there were no cases of human psittacosis from October, 1932, when the lid was clamped down to February, 1933.

Suddenly, however, in the spring of 1933 there occurred five cases in Los Angeles County in rapid succession, four of which were fatal, all traceable to different sources of infection. Later in the year psittacosis outbreaks occurred in Maine, Connecticut and Hawaii. However, it can be said to the credit of the California health authorities, that all cases occurring outside of California during 1933 appear to have been contracted from birds that were shipped from California in violation of regulations of the State Department of Public Health. As a direct result of these irregularities which resulted in an order from the Acting Secretary of the Treasury of the United States, the California State Board of Health on February 10, 1934, ruled that in the future "no shipping certificates will be issued without a report from a laboratory duly designated for that purpose showing that 10 per cent of the shell parrakeets have been examined by autopsy and laboratory tests on mice and found to be free from psittacosis infection, and further that no bird under eight months of age be offered or accepted." A branch of the State Laboratory was immediately established in Los Angeles and work was begun.

In the latter part of March of this year, the Pittsburgh outbreak of over twenty-five cases with eleven deaths occurred which was charged to California parrakeets. This, of course, demanded drastic action; so on April 2, the director of the State Department of Public Health issued an order to the effect that shipping permits both for interstate and intrastate movement of parrakeets be discontinued.

Thus we see the lid has again been clamped down tight, and we have reason to believe it will remain so until such time as state and federal authorities complete control measures for adequate protection of public health.

The previous procedure, based on a system of segregation and inspection, while helpful, was inadequate as evidenced by the continuance of cases here and in other states. The recent plan of February 10, 1934, which provided for a 10 per cent laboratory test of all aviaries before certification, was regarded as insufficient. Therefore, a more stringent procedure with additional safeguards was formulated and adopted on April 24. This plan provides for a 10 per cent double laboratory check of aviaries before breeding is permitted. Then the young birds are put in separate pens and, when eight months old, are subjected to a 10 per cent laboratory test and, if found free from infection, are banded, coded and released. Affidavits have to be signed by the aviary owners to follow outlined procedure. All shipments have to be counted, and code numbers checked and many other details carried out. The program seems promising, provided sufficient personnel can be secured for its rigid enforcement. If not, there would seem to be but one alternative and that is complete extermination of psittacine birds, and disinfection of premises and equipment.

Then, if the public still demands parrakeets, let the breeders start anew with clean stock. Many public health authorities believe the latter method would be cheaper and better in the long run. We all know how hoof and mouth disease was eradicated from the dairy herds of California. The psittacosis problem seems comparable. This procedure was strongly advocated by our most eminent authorities over two years ago and, had it been followed, the industry would now be restored, and there would have been a saving of many human lives.

PLAGUE*

By W. H. Kellogg, M. D.
Berkeley

Discussion *by K. F. Meyer, Ph.D., San Francisco*

THE history of plague as one of the five great epidemic diseases is too well known to require more than passing reference. The black death of the Middle Ages, responsible as it was for probably 25,000,000 deaths in a single century; the plague of Florence, for knowledge of which many are indebted to the Decameron of Boccacio; and the great plague of London are interesting historically, and as chapters in the history of medicine, but it is not epidemics of this kind that we fear at the present time.

Our very thorough knowledge of the cause and mode of spread of this disease insures us against such devastating outbreaks as the 1663-65 plague of London; although the cost of controlling epidemics once they are started runs into the millions for any given community, as two of our California cities know to their sorrow. Probably the traditional ounce of prevention looms larger in importance in this disease than in most any other, for its control depends, not on individual effort as in smallpox, but on community measures. These measures are not the usual ones of general sanitation of the environment, which are so effective in controlling cholera, typhoid and yellow fever; but

* From the State Hygienic Laboratory, Berkeley.

* Read before the General Medicine Section of the California Medical Association at the sixty-third annual session, Riverside, April 30 to May 3, 1934.

they require a specialized form of sanitation not invoked except for plague, and which is very expensive in its application. The outstanding epidemiological characteristic of plague, and one which it is well to remember, is its habit of recurring in epidemic form at intervals which are unusually long, measured in decades, even centuries. After these long periods of quiescence, during which it lies dormant in some endemic wild rodent focus, it takes on, for unknown reasons, renewed activity, spreads to the rat population of nearby communities, and thus is off on another world-wide pilgrimage, or pandemic. The pandemic spread can doubtless be ascribed principally to the traveling propensities of the rat, but not altogether. In other diseases, not spread by rats, we see the same pandemic tendencies.

APPEARANCE OF PLAGUE IN AMERICA

We are now in the midst of a pandemic of plague which began about 1894 in China, and which has wreaked its greatest havoc in India, where the deaths in 1903, 1904 and 1905 reached nearly a million a year. Until the present outbreak, plague had been absent from Europe for 200 years, and it had never visited the Americas; but in 1900 it reached San Francisco, and, at about the same time, Santos, Brazil. I was bacteriologist for the Board of Health of San Francisco at the time, and it was my fortune, or misfortune as it seemed for some time thereafter, to discover the first case in the person of a Chinese, who died of some mysterious disease after a very brief illness. My report to the board precipitated a chapter of medico-political events that almost pass the bounds of credibility. The story of the experience of San Francisco and its Board of Health in this, the first epidemic of plague in the western world, has been told elsewhere,[1] and I can only refer to it briefly at this time. It was a remarkable chapter, involving as it did a struggle between a conscientious board of medical men of the highest type against the malign attack of all the influences that feared undesirable publicity for the City of San Francisco; that feared loss of business, or had political reasons of various kinds, and of individuals, some of them in the medical profession, who yearned for publicity. These influences were successful, in the general ignorance of the disease, both lay and professional, in securing the partisanship of supervisors (but not the Mayor), of many members of the medical profession, including some prominent in medical educational circles, of judges of municipal and of federal courts, of legislators, including the Governor of the state, who was particularly active and bitter in the campaign against the local Board of Health. The activities of this opposition extended even to federal officials of note—the Surgeon General, and the Secretary of State, and they even enlisted the aid of the President himself. But we lived through it all, the Board held steadfast. The number of deaths in this first epidemic totaled 113.

1 Kellogg, W. H., Present Status of Plague with Historical Review, Amer. Jour. Pub. Health, Nov. 1920.

RÔLE OF THE RODENT IN PLAGUE

Plague has apparently established a permanent endemic focus among the ground squirrels of California, the menace of which, to be appreciated, requires a brief consideration of the habits of plague. The plague bacillus belongs to the hemorrhagic septicemia group, most of the members of which are pathogenic for the lower animals only. Plague, or bubonic plague as it is generally called, is primarily a disease of rodents, and only secondarily a disease of man. During interepidemic periods it lies dormant in low grade infections prevailing among wild rodents in certain localities which hitherto have been confined to Asia. From these foci it spreads, when the time is ripe, to that ubiquitous domestic rodent, the rat, and so to man. The means of spread from rat to rat, and from rat to man, but not from man to man, is the flea, which, gorged with blood teeming with bacilli taken up during the terminal septicemia of a dying rat, deserts its host with the onset of the chill of death and seeks a new host. It matters not to ceratophyllus or cheopis if the first available prospect is a human instead of a rat. In the meantime the plague bacilli are packing his proventriculus so that with attempts to feed again, a regurgitation occurs and virulent plague bacilli are deposited on the skin in the vicinity of the atrium of the bite, ready to be rubbed in with scratching. Plague bacilli do not infect the flea, but they may remain alive in his interior for as long as seventy days.

BUBONIC PLAGUE

Plague in man contracted in this way results in the bubonic form characterized by the development of the bubo, extremely painful, especially to touch. The bubo, usually only one, is more often than not in the groin below Poupart's ligament. The group of glands at the saphenous opening seems particularly likely to be involved, and there is no evidence that this is due to inoculation of the extremity. The buboes do not go on to suppuration excepting in cases that end in recovery. When death occurs it is usually within three or four days, or perhaps a week, and at postmortem the bubo is seen to consist of swollen lymph glands matted together with an abundant edema and hemorrhage. Section through the mass shows the cut surface of the individual glands to present, because of hemorrhage and necrosis, a purplish mottled appearance. The glandular involvement that is so characteristic of the disease is not always in the form of a visible bubo, but may be concealed in the abdomen, or elsewhere.

The definite diagnosis is made by puncture of the bubo of a suspected case with a small needle and syringe (the hypodermic is too small), the bloody fluid aspirated being smeared on a glass slide for microscopic examination, planted on culture media, and inoculated into guinea pigs. (The State Laboratory at Berkeley should be notified immediately of any suspected cases.) A case of plague of the bubonic type is not contagious in the ordinary sense, and not much of a danger to associates, at least in average white populations, for the reason that, even though fleas abound, a

septicemia is not common and the transfer of fleas from person to person is relatively infrequent. The flea to be feared is the rat flea which has been forced to seek a new host. Quarantine of persons, therefore, is unnecessary, but hospitalization of cases, and the destruction of rats and insects in the house where the infection occurred is necessary.

PNEUMONIC PLAGUE

Pneumonic plague is another matter altogether. Although caused by the same bacillus and having its primary origin in rodents, it is so different clinically and epidemiologically as to be, to all intents and purposes, another disease. Sometimes, in the course of an attack of bubonic plague, particularly when the lymph glands involved are in the neck or axilla, a secondary pneumonia caused by the plague bacillus develops. Isolated cases of this kind occur in bubonic outbreaks, but do not ordinarily lead to a spread from person to person of the pneumonic form. There are, however, circumstances which determine the development of an organ virulence on the part of the prevailing strain and then a pneumonic epidemic is imminent. The determining influences are little understood, but it seems likely that the rodent origin and, also, climatic factors play their part. Plague in ground squirrels and similar species, such as the tarbagan of Manchuria, frequently affects the lungs of these animals but rarely does so in rats. Guinea-pigs inoculated from plague-infected rats do not show pneumonia, but they frequently do when inoculated from ground squirrels, showing a difference in the strains. The pneumonic plague outbreaks that occurred in Manchuria in 1910-11 are said to have originated among trappers of the tarbagan, the animal that maintains the permanent focus in that country. In California, plague reached the ground squirrel population soon after its first appearance in San Francisco, the contact between rat and squirrel probably taking place in the vicinity of the Port Costa warehouses.

In Oakland in 1919 there occurred an outbreak of pneumonic plague originating in a case of plague pneumonia that was secondary to an axillary bubo, the man having been squirrel hunting immediately before the onset of his illness. A total of thirteen cases occurred in rapid succession, including two physicians and two nurses, and resulting fatally in all except one. The reason for the limitation of spread in this small group is not clear, but it may be that prompt hospitalization of all cases had something to do with it.

A similar outbreak occurred in Los Angeles in October, 1924, with a total of thirty-two cases, all fatal except two. This series began in a house occupied by ten people in the Mexican quarter. Nine of these died, as did sixteen relatives and friends who visited the house during the illness of the first patient. Other contacts to develop the disease and die were a nurse, a priest and an ambulance attendant. The quarter was placed in quarantine, and patients sent to the county hospital where a plague ward was established and every precaution taken to protect attendants. Mysteriously, as with the Oakland epidemic, new cases suddenly ceased.

Perhaps, in this connection, the observations of Teague and Barbour in Manchuria offer a clue. These observers noted that the most obvious difference in conditions between Manchuria, where fifty thousand deaths from pneumonic plague occurred, and India, where, among hundreds of thousands, all were of the bubonic type excepting about 2 per cent, and no epidemic prevalence of the pneumonic type ever occurred, was one of temperature. In Manchuria, during the epidemic the temperature was in the neighborhood of 30 degrees below zero, centigrade, and in India 30 degrees above. Teague and Barbour carried out animal experiments which showed that the plague bacilli contained in fine droplets of pneumonic sputum would die from drying in a few minutes unless they were suspended in an atmosphere with an extremely small water deficit. Such an atmosphere is common in cold climates and uncommon in warm ones. If this is the explanation, we in California may extract such degree of comfort from it as is possible in face of the fact that we have at our doors one of the world's permanent foci of rodent plague. As to the inhabitants of our eastern states, the outlook is little, if any, more hopeful, for it is easily possible for a person in the incubation period to journey to New York or Chicago in the winter where he would find the Manchurian conditions to perfection. Should he then develop a plague pneumonia, the consequences would be terrible to contemplate.

DIFFERENTIAL DIAGNOSIS

Physicians in California should be on the watch for possible cases of plague. The bubonic form, when not suspected, is likely to be mistaken for typhoid or some pyogenic infection. An acute solitary bubo, tender, but with little or no external inflammatory appearance (on the contrary the skin frequently has a blanched look) is suspicious. The most recent case of plague in California was in the person of a storekeeper in the town of Whittier, who died with a femoral bubo on one side only last August. No source for his infection was ever traced. Pneumonic plague cases are not markedly different clinically from other pneumonias. The onset is abrupt and prodromes are rare. Chilly sensations are complained of, but a rigor is not common. The patient has headache, fever, anorexia, and an increased pulse rate. Within 24 to 36 hours the temperature goes to 103 or 104 degrees, and cough with dyspnea develops in the same time. The expectoration is at first scanty and consists of mucus which soon becomes blood tinged. Later it becomes thinner and of a bright red color. Plague bacilli are numerous in the sputum at this stage. The typical rusty sputum of croupous pneumonia is not observed. The course of the disease is short and the mortality almost one hundred per cent. In its pathology it is a wet lobular pneumonia resembling very much the pneumonia seen accompanying epidemic influenza, with which disease the first few cases are almost sure to be confused. The cut surface of the lung shows lobules of consolidation well circumscribed, of a bluish red appearance, the tissue immediately surrounding being edematous and exuding large

amounts of thin red nonpurulent fluid. In patients that live a little longer, the areas have a characteristic rose gray color, are finely granular, and are characterized by the absence of fibrin. The areas of consolidation vary from 1 to 6 centimeters, coalescing in older processes into pseudo-lobar areas, the borders of the original lobulation still being faintly discernible. In Manchuria, it was a common observation that death occurred from an overwhelming septicemia before consolidation developed, the lung showing only an inflammatory engorgement. Under favorable atmospheric conditions, pneumonic plague is the most highly infectious and most rapidly fatal, as well as the most certainly fatal of all epidemic diseases. One should not knowingly risk a moment's exposure without the protection of a mask, and a plague mask is not the light gauze affair of the operating room, or of the public hysteria of an influenza pandemic. Such masks may be a protection to the field of operation against the comparatively inocuous pyogenic organisms that may be sprayed by the mouth of the surgeon, but they are worthless against plague and influenza. A properly constructed plague mask, as proven out in Manchuria, is made of a closely woven cloth and covers the entire head with a glass or mica window for vision.

SUMMARY

I have made this general discussion of plague as short as possible, including only the barest essentials of the epidemiology, in preparation for the final summary of the plague situation which is as follows:

Plague is now present in many parts of the world, it having become pandemic in the nineties of the last century. The last reports of prevalence in the United States Public Health Reports show plague present in British East Africa, Ceylon, Manchuria, four provinces of China, Dutch East Indies, Ecuador, Egypt, India, Indo-China, Iraq, Libya, Union of South Africa, Argentina, Bolivia, Madagascar, Peru and Senegal. Plague infected rats are being found in Hawaii. In India, the cases are running continuously at the rate of three to five thousand per week. Whether or not the pandemic is subsiding or only following the usual history of this disease which is to smolder along for twenty or thirty years or even for a century, then suddenly start on the upgrade again, no one can say. The certainties we have before us are that the disease is enzoötic in California, and that until governmental measures of extermination of the ground squirrels are prosecuted with the end in view of plague eradication, and not merely relief from the destruction of field crops, we have an ever present menace in our midst. The particular danger is to the people of eastern states rather than of the Pacific Coast, for the reason that pneumonic plague probably will not prevail extensively in the climate of California, but it is a very real menace in the climatic setting of the eastern seaboard in winter, to which locality it is easily possible for an incubationary carrier to travel. The State Board of Health is doing all that is possible to be done with the limited funds at its disposal. The real responsibility, however, would appear to be with the national government, since the danger is to the entire country.

Practicing physicians can have little part in the prevention of plague or, indeed, in curing cases, for vaccines and serums, while obtainable, are not of a high order of potency. Physicians should be able, however, to be of the utmost help in molding public opinion for the support of public preventive measures, and in advising people as to the danger of handling ground squirrels, and of exposing themselves to the possibility of picking up fleas in the neighborhood of squirrel burrows. Physicians should also, it goes without saying, be on the alert for sporadic cases of plague, and should not forget the possibility of plague when considering the diagnosis of influenzal pneumonia.

State Hygienic Laboratory.

DISCUSSION

K. F. MEYER, Ph. D. (Hooper Foundation for Medical Research, San Francisco*).—In order to discuss the various subjects here presented, I prepared a table in which the principal transmission chains are illustrated by concrete examples on certain known or incompletely studied diseases of animals and man. The first part of the tabulation deals with the infectious diseases which may be conveyed from a warm-blooded animal, either by the percutaneous, the alimentary or the permucosal routes to man. The name of the disease is followed by the species of animals which serves as the principal "reservoir" or "spender" of the respective infective agent. This grouping, although it makes no claim for completeness, comprises most of the known infections, but not the parasitic diseases which are generally described under the name of "zoönoses." In order to avoid any unnecessary confusion, the listing of the bacterial invader or virus is omitted. The special discussion will compensate for this incompleteness. Since this group comprises infections that are exceedingly variable with respect to their localization, portal of entry, mode of transmission, biologic characteristics of their disease incitants, etc., it is naturally impossible to give general criteria. However, the following features are worth recording: (*a*) The majority of the microörganisms, or viruses, responsible for the diseases possess broad "infection spectra." In other words, they are capable of infecting in nature a great many animal species, as well as experimentally established hosts. For example, spontaneous anthrax has been observed in man, horse, ox, sheep, pig, goat, buffalo, camel, wild game animals and carnivora in zoölogical gardens; also in the hare, dog, cat, guinea-pig, chicken, duck, goose and ostrich. Experimentally the bacillus is transmissible to mice, rabbits, guinea-pigs, rats, marmots, dormice, pigeons and occasionally to frogs, seahorses, etc. In this connection, one also recalls that in nature rabies is confined to a comparatively small number of species (dogs, cats, coyotes, foxes, wolves, jackals, horses, cattle, pigs, deer and moose), but artificial transmission is possible among a wide range of animals. In fact, it may be assumed that no mammal nor even bird is insusceptible.

(*b*) The chains, as a rule, are broken following the first transmission to man. No spread of the infection results from the exposure of a susceptible animal or man to the human being who contracted the zoönosis. In a few diseases, a restricted mode of natural transmission—percutaneous introduction of the virus through a bite wound, as in rabies or rat-bite fever—is in part responsible for this behavior. On the other

* Dr. Karl Meyer's discussion took into consideration the other papers in a symposium on "Animal-Borne Diseases," which were read before the Section on General Medicine at the annual session held at Riverside.

hand, the causative organism must maintain itself through continuous passage in the animal host. A change of host, as for example, a transfer to man, will sooner or later lead to a blind ending of the chain. In this general formulation, the statement, that the passages are ultimately broken, applies without restrictions to the entire group.

(*c*) In a few of the infections, the clinical and anatomical character of the disease in man is identical with that of the animals. A change of host does not alter the principal diagnostic signs. Some authors have pointed out that the infections due to the Bang's bacillus present a noteworthy exception. In man, it manifests itself as a septic fever, while in cattle as a localized process in the genital tract or udder. It is not unlikely that these differences are only relative. Very little is known concerning the early clinical course of a Brucella abortus infection in cows. According to Thomsen, cattle, and according to van der Hoeden, horses infected with the Bang's bacillus may have fever of an undulating type. The comparative clinical study of the entire group deserves further detailed scrutiny before generalizations are in order.

The second part considers the transmission by an animal of a low organization, in variably poikilothermic, either a flying insect or a nonflying arthropod such as a tick, flea or mite. The disease incitant localizes and multiplies in the poikilothermic host. All the latter are not merely "vectors or transmitters," since it is well known that only certain species of insects, ticks or mites are capable of conveying the disease germ. Between the causative agent of an infection and the poikilothermic host a specific mutual relationship is thus established, and the word "vector" is merely retained for the sake of convenience in order to avoid lengthy circumscriptions.

The infection chains of this nature reveal a well regulated alternating organization. Man and insect continuously interchange their position. The infection is, with two exceptions, never transmitted from man to man, nor is it spread from insect to insect. As a rule, the transmission: man-insect-man-insect frequently induces, with the alteration in the host, a change in the life-cycle of the parasite. To be sure, the details for such a life-cycle are only known for the malarial parasites, and it remains for future investigators to determine the existence of similar conditions for the trypanosomes, the rickettsias, the viruses, etc. The size of the parasite, and the lack of proper morphologic differentiation, may offer unsurmountable difficulties in this direction. One is justified, therefore, in assuming a transition in the life-cycle, only provided the experiment indicates that the transmitting insect is unable to convey the infection immediately after the intake of the disease producing agent. If, however, a few days elapse and if the "ripening process" is controlled by the temperature and the humidity of the environment, and infection is conveyed, it is then most likely that the parasite has undergone not merely a process of migration (from the digestive tube to the salivary or other glands), but a considerable multiplication. Evidence along these lines has not only been forthcoming for the microscopic, but also for the ultramicroscopic disease incitants. However, it is impossible, with the exception of malaria and trypanosoma, to prove that the exogenous multiplication (that is, in the insect) differs quantitatively from the endogenous development in man. Doubtless, the continuous change between vastly different hosts must be accompanied by profound alterations in the biologic activities of the parasites. These are indicated by special sexual phases in the life-cycle, and by organotropic properties which differ vastly from those observed in man or animals. This behavior is, to a certain extent, self-explanatory, but not readily comprehended is the observation that the *insector vector is very slightly, if at all, affected by the infection.* The life span is neither shortened, nor is the nutrition, reproduction or locomotion impaired despite the extensive changes which the parasite may produce in the digestive tube or in the sex organs (spirochaetes in ornithodorus). For the preservation of the parasite, this behavior is of the greatest importance. Should the vector succumb to the infection, or should the desire to suck blood disappear, then the transmission to man would cease and the chain would be regularly broken in the insect. In due time, the disease would become rarer and finally disappear entirely.

A study of subgroup 3 to 5 reveals further that the disease incitants are not only infectious for man, but can be inoculated experimentally into various species of mammals. One must, therefore, consider the possibility that in the natural transmission chains, man may be substituted by other homeothermic hosts. Or in other words, animals may serve as "virus reservoirs" from which the vector may draw the infective agent, and then transfer it to man. The existence of these conditions is of the greatest consequence for the epidemiology and control of certain disease entities.

In order to analyze these possibilities, it is necessary to determine (*a*) that the respective infection occurs spontaneously in higher animals, since not every experimentally proven host is also a natural host; (*b*) that a transmitter exists which sucks blood from man as well as from the suspected animal species; and (*c*) that epidemiologic observations conclusively prove the transfer of the disease from the animal to man through the agency of the hematophagous vector. If one sifts the available results of research from these points of view, one may find the following examples:

1. In dengue fever, yellow fever, sand-fly fever, African sleeping-sickness, five-day fever, Carrion's disease or verruca peruana, only one homeothermic natural host, man, is known. There are inconclusive reports available which would indicate trypanosomes in many respects to be similar to the trypanosoma gambiense occurring in sheep, goats, antelopes and crocodiles; but their relationship to human sleeping-sickness is by no means clear.

2. Animal reservoirs have doubtless been established for the relapsing fever "spirochaete" in burrowing rodents, and even in larger animals. Their epidemiologic importance is considerable and deserves detailed investigations. By no means explained or evaluated are the findings of the malarial parasite in anthropoid apes, the schizotrypanum cruzi in monkeys, opossums, cats and armadillos, or the presence of the tsutsugamushi rickettsia in mice.

3. The reservoirs in different mammals, as important virus sources for the transmitting arthropod, are proven for the following diseases:

Rocky Mountain Spotted Fever.—Dermacentor venustus as common vector; wild rodents serve as reservoirs.

Tularemia.—Dermacentor venustus, chrysops discalis (fly). Animal reservoirs: wild rabbits, squirrels, water rats, wild rats, woodchuck, lemming, field mice, etc.

Plague.—Various species of fleas, in particular Xenopsylla cheopis. Reservoirs: rats, squirrels and tarabagans.

Probably the leishmanioses could be classed in this group, but the final word has not been spoken concerning the transmitting insect, whether it be a Phlebotomus or another arthropod.

In every respect, it is a noteworthy fact that the infections just mentioned make their appearance in man only when their distribution in the animal reservoir assumes epidemic proportions. The remarkable, and well regulated dependence of bubonic plague on the simultaneous or preceding high mortality of rats from spontaneous plague is a well known epidemiologic fact. The causative bacteria exist and multiply principally in the mammalian hosts, and man acquires the disease merely by accident. In this respect, the maladies resemble those of the first group; with the difference, however, that the transmission from animal to animal, and the transfer from animal to man is never direct, but dependent on the poikilothermic blood-sucking vector. Yet, this rule is not applicable to tularemia and plague.

Authentic observations leave no doubt that plague may be transmitted from rat to rat, or in the form of

pneumonic plague from man to man, without the intervention of the flea. The contact transmissions of tularemia from rabbits to man, through the crushing of ticks or handling of cultures, are well known. It may not be mere coincidence that these two maladies are the only insect-borne infections caused by disease incitants which definitely belong to the bacteria. The microörganisms are very closely related, and it is the bacterial character of the disease incitant which finally controls the highly contagious character of tularemia and plague. To this, one may further add the fact that no particular portal of entry is required for the penetration of the bacteria. They invariably produce a septicemic penetration of the tissue of the host leading to a liberal elimination of the microörganisms in the pathologic discharges.

SARCOMA OF THE TESTICLE

By THOMAS A. CARD, M. D.
AND
ELMER M. BINGHAM, M. D.
Riverside

DISCUSSION *by Oran I. Cutler, M. D., Loma Linda; E. Forrest Boyd, M. D., Los Angeles.*

TUMORS of the testicle are rare when compared to new-growths in other parts of the body. Morris [1] states that less than 3 per cent of malignant growths involve the testicle. Young [2] gives figures from the Brady Urological Institute, where there were twenty-five tumors of the testicle in twelve thousand urological cases (0.2 per cent). The incidence of occurrence is given [3,4,5] as one in two thousand male admissions.

Tanner [5] states that six hundred cases had been reported in 1922. Sarcoma is by far the rarest; in fact there has been, and is, considerable disagreement as to the diagnosis of sarcoma of the testicle. Bell [6] believes many have been, and are still wrongly diagnosed. All of the cases formerly reported are not accepted. Under the newer classification many of the former cases of sarcoma fall into the seminoma group. Cairns [7] states that lymphosarcoma is the only one of this group which has not been proved of teratomatous origin; he had encountered three cases. Thus, while recent figures give the incidence of sarcoma testis as 0.4 to 1 per cent, we find a series of sixty-one cases by Coley [10] in 1915 in which the incidence was 77 per cent, and Kober's [11] series of 114 sarcoma cases in 1899. In contrast to this, Kelly [9] quotes Rubaschow as finding only twenty-four cases in the literature (1926); Hinman,[12] twenty-two cases with no sarcoma; Higgins,[13] twenty-three cases with four sarcoma, two of these diagnosed only clinically; O'Crowley and Martland,[14] two cases of lymphosarcoma in a series of thirteen; and Tanner, one hundred cases with no sarcoma; they are not included in his classification of testicular malignancies.[5]

Neoplasms of the testicle may occur at any age from less than one to eighty years. Sarcoma may occur at any age, the average being thirty-five years. Dew [15] reports one case, a man of eighty-four, with rapidly growing sarcoma of both testes, probably metastatic. One case in Kober's series was eighty-one; 14 per cent were over fifty. In Tanner's series, 12 per cent were over fifty.[5]

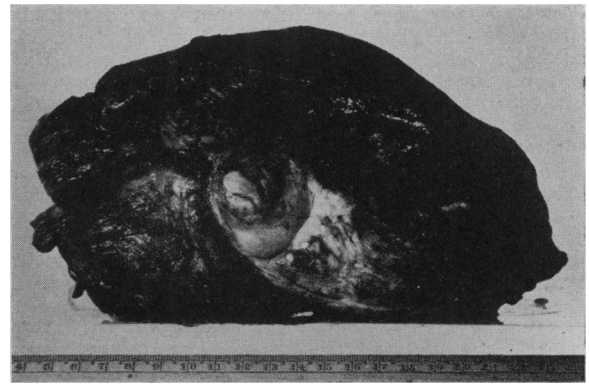

Fig. 1.—Tumor removed at operation.

While rare in occurrence, testicular malignancies carry a high mortality. Most patients have metastasis, not always demonstrable, when examined. In Higgins' [13] series the average lapse of time between appearance of tumor and examination was ten months. The tendency to spread by the vascular system is marked. Simons [19] states that a few—the genuine sarcomata—spread only by the vascular system. Those cases still alive four years after treatment are few, regardless of the type of malignancy. The average mortality given is 80 to 95 per cent, the sarcoma cases being in the high mortality group.

Two opinions are expressed regarding methods of treatment. Coley advocates simple removal of the tumor and administration of bacterial toxins. Tanner believes that a radical operation with removal of the lumbar lymphatic gland is more beneficial than simple castration followed by radiation or toxins. The operative mortality is 10 to 20 per cent. Hinman [17] reports five consecutive radical operations without operative mortality. Hinman, Higgins, Keyes [16] and Peirson [18] recommend surgery with radiation preceding or following the operation. Some recommend the x-ray both before and after surgery. It would seem that any operation, radical or simple, would be inadequate. It is a physical impossibility to remove all of the lumbar lymphatic tissue. Surgery will not reach the hematogenous metastasis. Surgical treatment of sarcoma in any part is very unsatisfactory except when seen early. With recent development in high voltage therapy, greater accomplishments should be expected from simple surgery supplemented by radiation.

REPORT OF CASES

CASE 1.—C. L., an Indian male of eighty-three, was admitted to the Riverside County Hospital, November 9, 1932. He complained of a growth in the left side of the scrotum, first appearing three years previously and increasing steadily in size. Further history was unsatisfactory. Examination showed a large, well-nourished Indian male. Teeth had all been removed. Heart and lungs were fairly normal. Blood pressure was 190/100. There were no abdominal masses. There was a solid tumor mass in the scrotum approximately eight inches in diameter, irregular in outline and not tender; the skin was not adherent to the mass. There was a surgical scar of a left herniotomy (?). The inguinal glands were not enlarged. Laboratory: Urine, trace of albumin and a few pus and blood cells; hemo-